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GUILLAIN-BARRÉ SYNDROME AFTER BRACHIAL PLEXUS TRAUMA

Case report

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ABSTRACT - The Guilllain-Barré syndrome (GBS) is an acute predominantly demyelinating polyneuropathy. In many cases GBS is preceding by infection, immunization, surgery or trauma. Although there are a few reports of GBS after head trauma, there is no report of this syndrome after brachial plexus injury. We report on a 51 years-old man who presented GBS fifteen days after a brachial plexus trauma. The polineuropathy resolved completely in a few weeks. We believe that GBS was triggered by the trauma that evoked an immune mediated disorder producing inflammation and demyelination of the peripheral nerves.

KEY WORDS: Guillain-Barré syndrome, polyradiculoneuropathy, brachial plexus, trauma.

Síndrome de Guillain-Barré após traumatismo de plexo braquial: relato de caso

RESUMO - A síndrome de Guillain-Barré (SGB) é uma polineuropatia predominantemente desmielinizante, que ocorre na maioria das vezes após uma infecção, vacinação, cirurgia ou traumatismo. Embora tenham sido descritos alguns casos após traumatismo crânio encefálico, ainda não foi referido caso de SGB após traumatismo do plexo braquial. Relatamos o caso de um homem de 51 anos que 15 dias após ter apresentado paralisia traumática do plexo braquial, desenvolveu SGB. Recuperou-se inteiramente em algumas semanas. Achamos que em nosso caso a SGB foi desencadeada pelo traumatismo, que provocou distúrbios imunológicos com conseqüente acometimento dos nervos periféricos.

PALAVRAS-CHAVE: síndrome de Guillain-Barré, polirradiculoneuropatia, plexo braquial, traumatismo.

The Guillain-Barré syndrome (GBS) is the most common cause of acute generalized weakness. Its incidence is 1-2/100000 people[1]. It is an acute inflammatory demyelinating polyradiculoneuropathy due to an immunological reaction directed at the peripheral nerves. The major clinical manifestation is an ascendant symmetrical weakness with reduced or absent tendon reflexes, minimal sensory loss and cranial nerve palsies. It may be preceded by infectious illness, immunization, surgery and trauma[2]. Although there were a few cases of GBS described after head injury[3,4], there is no report of GBS triggered by peripheral nerve trauma.

We reported a case that presented GBS syndrome after a trauma of the brachial plexus.

CASE

A 51-year-old man suffered a traumatic superior right brachial palsy after lifting a bar of 100 kg. Fifteen days after he felt weakness and had paresthesias in the four limbs that progressed in two days, and soon after he presented a bilateral facial palsy. There was a predominantly distal weakness in legs and arms and severe weakness of right biceps, deltoid, supinator longus, supraspinatus and infraspinatus with absent tendon reflexes and mild objective sensory loss in the four extremities. The cutaneous plantar reflexes were indifferent. There was a bilateral peripheral facial palsy. The other cranial nerves were normal. He had no radicular symptoms. His blood pressure was 130/80, pulse was 80/min, breath rate 22/min and he was apyretic.

The blood chemistry and hemogram findings were normal. Tests for *Campylobacter jejuni, Cytomegalovirus,* HIV, HTLV1 and herpes virus were negative. Spinal fluid examination showed a protein content of 158 mg/dL with 2 cells/mm^3. Nerve conduction study was performed 7 days after and revealed slowed motor conduction velocities with conduction block in some nerves (Table 1). The sural nerve action potential was absent but the others action potentials were normal (Table 2). On needle examination there was denervation in distal muscles of upper and lower limbs and in the muscles of the right superior brachial plexus.

In three days the clinical findings were steady, and we

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Table 1. Motor nerve conduction.

Nerve	DL (ms)	PL (ms)	DA (mV)	PA (mV)	NCV (m/s)	F wave (m/s)
L Median	3.6 (NV<4)	9.2	4.6 (NV>4)	2.9	41.8 (NV>52)	37.4 (NV<33)
R Median	3.6	9.9	4,8	1,8	38.6	38.7
L Ulnar	3.9 (NV<3,7)	7.7	4,4 (NV>4)	2,8	54,1	35.5
R Ulnar	3.8	8.3	5.2	3.2	46.9	44.8
L Fibular	5.8 (NV<3,7)	20.3	1.9 (NV>2)	0.4	28.8	0
R Fibular	6.2	17.6	2.6	1.9	33.9	0
L Tibial	5.4 (NV<3,7)	20.5	4.2 (NV>2)	0.8	25.5	66.4
R Tibial	6.7	21,7	4.1	0.3	30.8	0

L, left; R, right; DL, distal latency; PL, proximal latency; DA, distal amplitude; PA, proximal amplitude; NCV, nerve conduction velocity; NV, normal value.

decided to introduce neither immunoglobulin, nor plasma exchange treatment. On the day 10th, his movements began to improve and 20 days after he was walking with aid. There was a complete spontaneous recovery of the neuropathy in two months, although the brachial plexus palsy persisted unchanged.

DISCUSSION

The clinical and electroneuromyography picture of our patient fulfilled perfectly the diagnostic criteria for the typical form of GBS[2]. The literature is filled with factors considered as "triggers" in the GBS[1,2,5-7] about two-thirds of cases following a virus infection. The responsible viruses are Epstein-Barr virus, cytomegalovirus, other herpes virus, HIV, hepatitis A and B, rubella, mumps, influenza A, influenza B, Coxsackie virus and parvovirus. Among all bacteria described to be involved, *Campylobacter jejuni* and *Micoplasma pneumoniae* have been the most mentioned. Vaccination, surgery, epidural anaesthesia and drugs, have all been associated with some cases. There have been also reports of GBS associated to an underlying disease such as systemic lupus erythematosus, Hodgkin disease and sarcoidoisis.

There are only two reports of the disease appearing after head trauma[3,4]. The case reported by Vega-Basolto et al.[8] was of a man who presented head trauma three months before the surgery of a subdural haematoma. Thus, the GBS might be triggered by the surgery or the trauma. Although there was a case of recurrence of GBS after a lumbar decompression and fusion[9], and one following spinal root damage after epidural anaesthesia[10], our case is the first one describing GBS after brachial plexus injury.

Presently it is believed that both cellular as well as humoral immunities have a role in the physiopathogenesis of GBS[11]. There is a report of a lumbar nerve root injury inducing neuroimmune activation and neuroinflammation in the rats[11]. Another possible mechanism is the stress caused by trauma activating some latent or sub clinical process[4].

Table 2. Sensory nerve conduction.

Nerve	DL (ms)	A (mV)
L Median	3.0 (NV<3,4)	36 (NV>15)
R Median	3.4	40
L Ulnar	3.5 (NV<3,5)	37 (NV>10)
R Ulnar	3.8	31
L Radial	3.1 (NV<3,5)	14 (NV>5)
R Radial	3.3	13
L Sural	0	0
R Sural	0	0

L, left; R, right; DL, distal latency; NV, normal value; A, amplitude.

In regard to the reported case, it is hard to affirm whether actually exists a cause-and-effect relationship between the two entities or if it is a mere coincidence.

REFERENCES

1. Arnason B, Soliven B. Acute inflammatory demyelinating polineuropathy. In Dyck PJ, Thomas PK, Griffin JW, Low PA, Poduslo JF (eds). Peripheral neuropathy. 3.ed. Philadelphia: WB Saunders, 1993:1437-1497.
2. Asbury AK, Cornblath DR. Assessment of current diagnostic for Guillain-Barré syndrome. Ann Neurol 1990;27(Suppl):S21-S24.
3. Duncan R, Kennedy PGE. Guillain-Barré syndrome following acute head trauma. Postgrad Med J 1987;63:479-480.
4. Freitas GR, Freitas MRG, Ferreira MCL. Guillain-Barré syndrome and head trauma. Arq Neuropsiquiatr 1997;55:315-318.
5. Hughes RAC. Guillain-Barré syndrome and chronic inflammatory polyradiculoneuropathy. In Asbury AK, Thomas PK (eds). Peripheral nerve disorders. 2.ed. London: Butterworth-Heinemann, 1995:175-204.
6. Leneman F. The Guillain-Barré syndrome: definition, etiology and review of 1100 cases. Arch Intern Med 1966;118:139-144.
7. Ropper AH, Widjicks EFM, Truax BT. Antecedents and associated illness. In Guillain-Barré syndrome. Philadelphia: Davis, 1991:57-72.
8. Vega-Basulto S, Dominguez-Nápoles E, Infante J, Gutiérrez Munoz F, Debessa-Fernández R, Basultp-Barroso M. Hematoma subdural crônico y síndrome de Guillain-Barré. Rev Neurol 2004;3:1194-1196.
9. Ennis JH, Bednar DA. Lumbosacral fusion in a patient with recurring Guillain-Barré syndrome and acute brachial neuritis. J Spinal Disord 1992;5:217-218.
10. Steiner I, Argov Z, Cahan C, Abramski O. Guillain-Barré syndrome after epidural anesthesia: direct nerve root damage may trigger disease. Neurology 1985;35:1473-1475.
11. Rutkowski MD, Winkelstein BA, Hickey WF, Pahl JL, DeLeo JA. Lumbar nerve root injury induces central nervous system neuroimmune activation and neuroinflammation in the rat. Spine 2002;27:1604-1613.